Table 1. DISTRIBUTION OF TRANSFERRIN PHENOTYPES IN CHIMPANZEES

Phenotypes	Orange Park Colony Inbred	Orange Park Colony Jungle born	Boyer and Young group
AA	0	1	1
AC	3	2	3
AD	1	1	5
BC	3	2	4
CC	35	11	9
CD	16	0	1
DD	0	2	2
Total	58	19	25

were born in the colony, the latter being an inbred group of chimpanzees. CC was the most common of the phenotypes in both these groups and also in the chimpanzees analysed by Boyer and Young. However, phenotype CD, which appears to be rather rare in jungle born chimpanzees (putting our data and that of Boyer and Young together), was the next most common phenotype in the chimpanzees born at Orange Park. An original breeder female in the colony (Wendy No. 4) has been typed as CD, which accounts in part for the rather frequent occurrence of the CD phenotype in the inbred group. It should be emphasized that as larger numbers of chimpanzees are surveyed new phenotypes and new molecular forms of transferrin in addition to the seven phenotypes observed by Boyer and Young[1] and ourselves might well be discovered. Nevertheless, the use of a nomenclature which in effect describes the transferrin phenotypes of chimpanzees in terms of an allelic series of genes is not inappropriate as the following communication demonstrates.

We thank Dr. Melvin R. Sikov of the Department of Radiology, Wayne State University College of Medicine, for his advice on the radioactive iron-binding analyses and for generously permitting us to use his facilities.

MORRIS GOODMAN
ROSEMARIE MCBRIDE
EMILY POULIK
EDUARDAS REKLYS

Wayne State University College of Medicine,
Detroit.

Inheritance of Serum Transferrins in Chimpanzees

BY reviewing the familial relationships of chimpanzees born in the Orange Park Colony, the transferrin phenotypes of which were classified by the Boyer and Young scheme (preceding communication), it was possible to evaluate the genetic basis for the inheritance of these transferrins. The results are recorded in Table 1. To summarize the findings: of the twelve offspring of CC × CD matings, six were CC and six were CD. There were four offspring of AC × CC matings; three were CC and one was AC. The two offspring of BC × CC were BC and CC. The one offspring of CD × CD was CD, and the offspring of the CC × CC mating was CC. Thus without exception each offspring always had one major transferrin identical in type to one held by the mother and one major transferrin identical in type to one held by the father. Furthermore, there were fourteen offspring, each one having a transferrin phenotype which could be compared with the transferrin phenotype of only one of the parents; again, one major transferrin of the offspring was always identical in type to a major transferrin of the parent. Thus, as in the case of human transferrins[5] and rhesus monkey transferrins, these data support the hypothesis that a single autosomal multi-allelic series of genes without dominance controls the transferrin polymorphism of chimpanzees.

There would be four genes in the allelic series controlling respectively transferrins A, B, C and D. The minor transferrin bands would be related to the major bands, and their presence along with these major bands would be controlled by the same allelic system at a single locus. The appearance of the seven chimpanzee phenotypes

Table 1. TRANSFERRIN PHENOTYPES OF PARENTS AND OFFSPRING IN THE ORANGE PARK CHIMPANZEE COLONY

Name and No. of animal			Transferring phenotype		
Mother (No.)	Father (No.)	Offspring (No.)	M	F	O
Wendy 4	Bokar 5	Web 79	CD	CC	CD
Alpha 28	Hal 43	Halpha 176	CC	CD	CD
Bula 48	Hal 43	Easter 117	CC	CD	CC
Bula 48	Hal 43	Pix 157	CC	CD	CD
Bula 48	Hal 43	Hew 171	CC	CD	CD
Bula 48	Hal 43	Ball 195	CC	CD	CD
Bula 48	Jent 71	Bent 165	CC	CD	CC
Helene 62	Hal 43	Alec 139	CC	CD	CC
Soda 12	Hal 43	Lad 119	CC	CD	CC
Soda 12	Hal 43	Sadie 154	CC	CD	CD
Soda 12	Hal 43	Sally 78	CC	CD	CC
Fanny 102	Jent 71	Norma 166	CC	CD	CC
Gamma 58	Bokar 5	Margo 160	AC	CC	CC
Gamma 58	Bokar 5	Garbo 168	AC	CC	CC
Pati 42	Bokar 5	Beti 130	AC	CC	AC
Pati 42	Bokar 5	Portia 144	AC	CC	CC
Debi	Frans 103	Dena 186	BC	CC	BC
Debi	Frans 103	Chan 181	BC	CC	CC
Wendy 4	Jent 71	Went 143	CD	CD	CD
Fanny 102	Frans 103	Francy 224	CC	CC	CC
Wendy 4	(Jack)	Jenny 90	CD		CD
Wendy 4	(Jack)	Jed 67	CD		CD
Alpha 28	(Frank)	Alf 59	CC		CC
Alpha 28	(Frank)	Flora 100	CC		CC
Alpha 28	(Frank)	Alan 107	CC		CC
Helene 62	(Dick)	Dehn 95	CC		CD
Soda 12	(Frank)	Frans 103	CC		CC
(Fifi)	Bokar 5	Art 57		CC	CC
(Vert)	Bokar 5	Verb 97		CC	CC
(Vert)	Bokar 5	Kathy 146		CC	CC
(Coma)	Hal 43	Malcom 141		CD	CC
(Coma)	Hal 43	Polly 178		CD	CC
(Vert)	Hal 43	Val 169		CD	CC
(Banka)	Web 79	Wenka 170		CD	CD

Brackets denotes that the animal was no longer available for drawing serum due to death.

(Fig. 2 in the preceding communication) suggests that transferrins A, B and C each have one minor component migrating directly in advance of the major component, whereas transferrin D has two components migrating in advance of the major component, the slower of these more rapidly migrating bands occurring in the same position of the major component of transferrin C. Another genetic hypothesis to account for multiple transferrin bands in chimpanzees presumed to be homozygotic for the protein is that genes at more than one locus interact to control the molecular specificities of transferrin, with the observed phenotypes resulting from an allelic series at only one of the gene loci.

Our results by either genetic hypothesis support the assumption that CC is a homozygous phenotype and that AC, BC and CD are heterozygous phenotypes. As yet we have no information on the inheritance of phenotypes AA, AD and DD. Thus the assumption that the phenotypes designated DD and AA are homozygous in character still rests on very tenuous grounds. Clearly the genetic basis for the transferrin differences in chimpanzees cannot be thoroughly evaluated until a more extensive programme of breeding chimpanzees with these various transferrin phenotypes is carried out.

This work was carried out with support from grant *G*–14152 of the National Science Foundation, grant 226 of the National Multiple Sclerosis Society, and grant *H*–5691 from the National Heart Institute of the National Institutes of Health.

MORRIS GOODMAN
ARTHUR J. RIOPELLE

Wayne State University College of Medicine,
Detroit,
and
Yerkes Laboratory of Primate Biology,
Orange Park,
Florida.

[1] Boyer, S. H., and Young, W. J., *Nature*, **187**, 1035 (1960).
[2] Smithies, O., and Hiller, O., *Biochem. J.*, **72**, 121 (1959).
[3] Giblett, E. R., Hickman, C. G., and Smithies, O., *Nature*, **183**, 1589 (1959).
[4] Buettner-Janusch, J., *Nature*, **192**, 632 (1961).
[5] Parker, W. C., and Bearn, A. G., *Ann. Hum. Genet.* (Lond.), **25**, 227 (1961).
[6] Poulik, M. D., and Smithies, O., *Biochem. J.*, **68**, 636 (1958).
[7] Poulik, M. D., *Nature*, **180**, 1477 (1957).
[8] Goodman, M., and Poulik, E., *Nature*, **190**, 171 (1961).